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BALD EAGLE PESTICIDE RELATIONS

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Bald eagles have become scarce in many parts of the United States, and their reproductive success exceedingly low. They produced young in only 3 of 16 nests studied in 1964 in the mid-Atlantic States, and, on a nationwide basis, the percentage of young appears to be gradually declining (Sprunt and Ligas, 1963). Concern for conservation of the bald eagle led to the initiation in 1961 of a series of cooperative studies.

In this year, the National Audubon Society undertook a nationwide survey of numbers, distribution, and nesting success. At the same time, the Bureau of Sport Fisheries and Wildlife began investigations of the effects of environmental pollution on eagles.

The Bureau's first studies were focused on DDT. Experimental investigations of DDT toxicity to eagles and the metabolism of DDT by eagles were made in Alaska in the winters of 1961-62 and 1962-63. Residue analyses of eagles and eagle eggs were undertaken on a continuing basis. Beginning with eagles received early in 1964, analyses were expanded to include other chlorinated hydrocarbons. I will summarize the results and conclusions from those studies and briefly describe our current program and plans. Progress reports concerning portions of this work have been presented by DeWitt and Buckley (1962) and Buckley and DeWitt (1963).

DDT Toxicity Studies

The DDT toxicity studies were made to determine the dietary dosage of DDT that would kill eagles and hence to learn whether or not eagles were unusually susceptible to DDT poisoning. A second objective was to determine the quantities of residue present in the tissues of eagles killed by DDT, as an aid in understanding the importance of the quantities in eagles from the field.

Care, food consumption, and behavior have been described by Chura and Stewart (unpublished manuscript). Spermatogenesis in dosed birds has been discussed by Locke, Chura, and Stewart (unpublished manuscript). Hence these aspects need not be considered here.

The first experimental studies were performed with 11 eagles caught near the Chilkat River near Haines, Alaska, and kept at the Petersburg Experimental Fur Station. Dietary dosages were 5, 83,

414, and 2070 parts per million (ppm) computed on the basis of dry weight of the food, which consisted of ground salmon heads and other waste fish products. Toxicant added on a wet-weight basis was at the rates of 3, 48, 240, and 1200 ppm. The DDT (technicalgrade, p,p' isomer) was dissolved in vegetable oil and mixed thoroughly with the fish.

Two birds were fed clean food for the full 112 days of the study; two birds were fed at each of the three lowest dosages; and three birds were fed at the highest dosage.

Dosage was not continuously the same, for food consumption varied from day to day. The eagles on the highest dosage ate less from the start, and all eagles that died ate less as time passed.

During the first week of dosage, birds fed 5 ppm of DDT in their food consumed 0.3 mg DDT per kg of body weight per day (on the basis of their weights at capture). Those fed 83 ppm consumed 3 mg/kg/day; and those fed 414 ppm consumed 15-18 mg/kg/day. Two eagles fed 2070 ppm of DDT consumed 55-70 mg/kg/day, but the third consumed only 28 mg/kg/day during the first two weeks, less the first week (Table 1).

All eagles fed more than 400 ppm (dry weight) died in 2 months or less. Of the two fed about 80 ppm, one died in 71 days with pronounced tremors. The other survived 112 days but showed some

TABLE 1. TOXICITY OF DDT TO BALD EAGLES

Bird Number	Sex and Age	DDT Dosage: DDT Added to Diet[1] (ppm dry weight)	DDT Dosage: mg/kg Per Day[2]	DDT Dosage: mg Per Day[2]	DDT Dosage: mg Total	Days on Test	Died or Killed
1	♂ adult	0	0	0	0	112	K
10	immature	0	0	0	0	112	K
3	immature	5	0.3	1.4	120	98	escaped
6	♂ adult	5	0.3	1.2	38	77	D
2	♂ adult	83	2.8	15.8	730	71	D
9	♂ adult	83	3.2	16.0	1811	112	K
5	♂ immature	414	17.7	70.1	3173	62	D
7	♀ immature	414	15.4	87.0	3678	59	D
4	♂ adult	2070	70.0	331.6	5476	23	D
8	♂ immature-adult	2070	55.0	260.8	4631	18	D
4A	♂ adult	2070	27.6	110.3	1965	15	D

[1] Technicalgrade p,p' DDT was dissolved in vegetable oil and mixed with the diet of ground salmon heads and other fish. Dietary content of DDT expressed as parts per million of DDT in the wet weight of the food was approximately 3, 48, 240, and 1200. Computations to express DDT intake in terms of dry weight were made on the basis of an average moisture content of 42 percent, as determined in samples of the prepared food. Nominal dry weight doses reported by Buckley and DeWitt (1963) were preliminary computations based on an estimated 70 percent moisture content, the amount present in fresh fish fillets.

[2] Calculation of milligrams of DDT per kilogram of body weight per day is based on capture weights of birds. Daily intake of DDT is computed for the early dosage period before decline in food consumption by birds that died later. This period was March 9-15, 1962, for all birds except one. Bird 4A was started on dosage May 11 and ate little the first week; hence computations are for May 11-May 21, 1962.

tremors and erratic wing jerking suggestive of a toxic effect. Thus a dosage that would kill half the birds during a 3-4 month period may be near 80 ppm, possibly somewhat lower.

Mortality at this dosage level does not indicate any unusual susceptibility of eagles to death from DDT. In studies with other species at Patuxent, where DDT is similarly dissolved in oil and mixed with the diet, it has become evident that long-term tolerance limits for at least part of an experimental group are near 40 ppm for mallard ducks and 25 ppm for bobwhite and coturnix quail.

The one eagle that died on the 5 ppm dosage was believed to have succumbed from causes other than DDT. The other eagle on this dosage remained apparently well for 98 days, when it escaped. In studies the next year, 15 eagles were fed DDT in their diet at the rate of approximately 5 ppm. One died within 39 days, probably from causes unrelated to dosage. Four survived 120 days of dosage and 10, 60 days of dosage with no obvious ill effects.

Residue analyses were made by the colorimetric methods described by Schechter *et al.* (1945). Readings were made at wavelengths 590 and hence are primarily for DDT and DDD.

In the eagles fed the two highest dosages, where the cause of death almost certainly was DDT poisoning, the quantities of DDT and DDD in the brain ranged from 58 to 86 ppm (wet weight) (58, 63, 80, 85, and 86 ppm) (Table 2). These amounts were very similar to those associated with DDT-induced death in several other species of both birds and mammals, where average residues were 43-100 ppm, and a hazard zone could be considered to begin in the vicinity of 30 ppm (Stickel *et al.*, 1966).

The conclusion from the eagle toxicity tests is that there is little reason to suspect any unusual susceptibility of eagles to DDT mortality. The possibility of more obscure effects on physiology or behavior will require other studies.

DDT KINETICS

The second series of experimental studies, conducted in Alaska in the winter of 1962-63, was made to measure the storage and loss

TABLE 2. DDT RESIDUES IN BALD EAGLES KILLED BY DDT

Bird Number	Sex and Age	Dosage (ppm dry weight)	Brain	Liver	Muscle
6	♂ immature	414	63	250	73
7	♀ immature	414	80	—	291
4	♂ adult	2070	58	715	112
8	♂ immature	2070	86	391	169
4A	♂ adult	2070	83	—	149

[1] Colorimetrically determined by the method described by Schechter *et al.*, 1945. Read at wavelength 590, hence primarily DDT + DDD.

of DDT by eagles when the dietary intake was comparable to what might be available in the field. This information was sought as an aid in judging the likelihood that intake of small quantities of DDT over a long period of time would build up to lethal levels. A dosage of approximately 5 ppm dry weight was used; wet-weight equivalent was 3 ppm. Fish in some areas may contain much higher residues than these, and much lower in other areas. Residues in birds, sometimes eaten by eagles, also are variable.

The experimental design called for dosage of one group of birds for 60 days, one group for 120 days, and one group for 60 days followed by clean food for 60 days. Six eagles were in the 60-day group, four in each of the other two groups. Analyses were made for DDT and the metabolites DDD and DDE in various tissues and organs. Readings were made by gas chromatography following extraction in Soxhlet with petroleum ether and Skellysolve B, partitioning with acetonitrile, and passage through a florisil column.

In these experimental eagles, the amount of DDT + DDD in the tissues increased between 60 and 120 days on dosage and decreased after dosage was discontinued. A similar pattern of gain and loss followed in brain, liver, breast muscle, fat, and in the composited remainders (Figure 1). Rates were 0.4 to 0.8 per cent per day, computed as $\log_e$ (content at time 2/content at time 1)/ days. These rates were used to approximate the time at which half the toxicant would be gone, the time at which a steady state or equilibrium would be reached, and the residue level that would be reached at the time of equilibrium. Estimates were made as described by Nelson (1961) and by Doluisio and Swintosky (1965).

These procedures assume that the rates of loss remain constant with time. There is evidence, however, that this may not be strictly true, and that rates may decline as residue content of tissue declines (Bovard *et al.* 1961). Hence, the estimates of half-life derived here for DDT residues in eagle tissues may be somewhat short. Rates were estimated from the parts per million of chemical in the lipid (fat) portion of the tissues.

It was estimated that about half the residues of DDT + DDD would be gone in 3-5 months, and hence that a balance between intake and loss probably would require 1½ to 2½ years if this same dosage continued.

Brain residues of DDT + DDD reached 0.28 ppm (wet weight) after 120 days on dosage. The estimated level at equilibrium was less than 1 ppm, far below the 58-86 ppm found in eagles killed by DDT. Liver residues of DDT + DDD were 0.68 ppm after 120 days on

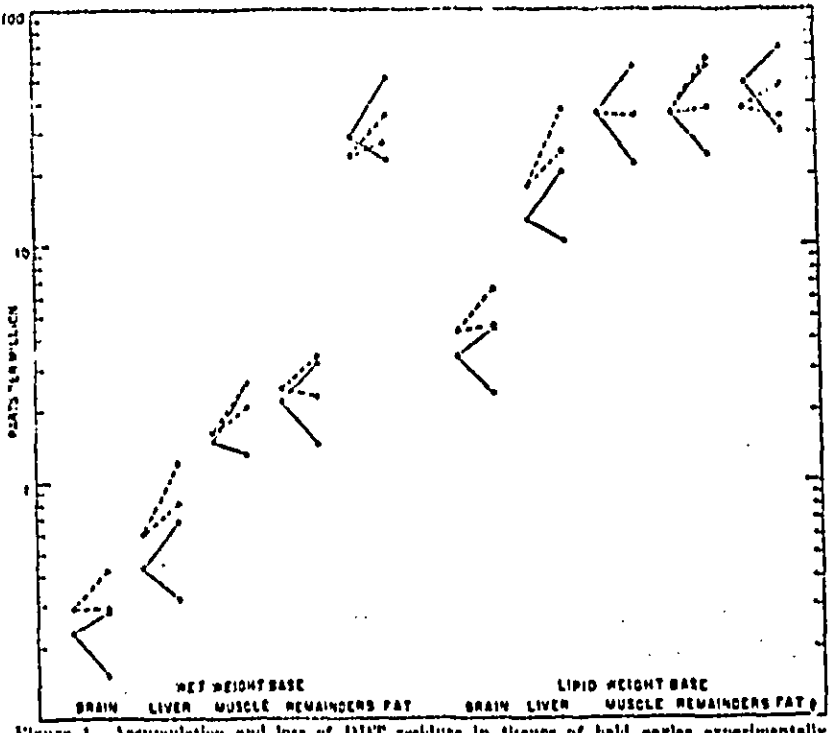

Figure 1. Accumulation and loss of DDT residues in tissues of bald eagles experimentally fed DDT. *Residues after 60 days on a diet containing 5 ppm p,p' DDT are shown at the left apex (averages for 6 birds).* Residues after 120 days on this same dosage are shown by the upper arm; residues after 60 days on dosage plus 60 days on clean food are shown by the lower arm (averages for 4 birds each). DDT plus DDD is indicated as a solid line, DDE as a broken line. *Coefficients of variation for the wet weight values were:* 20-29 percent for brain, liver, fat, and remainders and 40 percent for muscle. For the lipid weight values, coefficients of variation were 25-29 percent for brain, liver, and remainders and 36 percent for muscle and fat.

dosage. The level at equilibrium was estimated at 2 ppm, also far below the amounts in eagles killed by DDT.

Residues of DDE, a less toxic metabolite of DDT, also increased in tissues between 60 days and 120 days of dosage. Clean food for 60 days, however, did not result in a diminution of DDE in the tissues, and quantities in the liver in fact increased. It is not unreasonable that DDT would continue to be transformed by the liver after dietary intake stopped, for a considerable supply of unchanged DDT still was present in the various tissues. A portion of the loss of DDT from the tissues after dosage can be accounted for by transformation to DDE. DDE itself presumably would be lost in time and there is no reason to expect that it would have reached toxic levels in the experimental eagles. Quantities of DDE measured

at 120 days were 0.42 ppm in brain, 1.22 ppm in liver, 2.58 ppm in muscle, 3.38 ppm in remainders, and 35.68 ppm in fat.

The importance of tissue residues of DDE is not understood. DDE did not appear to be critical in deaths of a group of experimental cowbirds we studied, nor in certain field studies. However, DDE in large amounts will kill birds and its residues could well be important at some presently unknown quantity. DDE is not used as an insecticide, but is produced from DDT by many living organisms. DDE in eagle tissues thus may have been obtained as DDE from the fish the birds ate, or it may have been produced by the birds themselves from DDT consumed, making the interpretation of its presence especially difficult.

Gradual loss of DDT from tissues of animals after dosage is discontinued has been shown in many other studies. For example, Laug *et al.* (1950) and Ortega *et al.* (1956) described loss of DDT in laboratory rats; Bovard *et al.* (1961) in cattle, and Durham *et al.* (1963) in rhesus monkeys. Noma and Benfield (1965) reported loss of DDT and DDD in chickens and also noted a post-dosage increase of DDE, as was observed in the eagles.

The conclusions from this portion of the study are that continuous intake of DDT by bald eagles in quantities as high as 5 ppm in the diet is unlikely to produce lethal amounts in the tissues. DDT content of the tissues will increase slowly for many months before a metabolic balance is reached, and will be lost slowly when intake of DDT is discontinued. The pattern and rates are similar to those in other animals, indicating similar basic processes.

ANALYSES OF EAGLES FROM THE FIELD

Pesticide residues in the tissues of eagles from the field provide a measure of environmental exposure. They can be useful in assessing hazard if experimental studies have provided the basis for interpretation. Such interpretive studies have been few, confined largely to DDT, and focused on the determination of lethal quantities. However, enough information is available concerning dieldrin to permit at least tentative judgments.

In the summer of 1965, we began broad spectrum analyses of eagles from the field. Readings were made by gas- and thin-layer chromatography after extraction with petroleum ether in Soxhlet apparatus, partitioning with acetonitrile, and elution through a florisil column. Sixteen bald eagles have been completed. DDT, DDD, DDE, and dieldrin have been found in all of them and traces of heptachlor epoxide have been found in many. Brain samples were available for 14. Quantities of DDT + DDD were 1 ppm or lower in 13

specimens, a magnitude similar to that of eagles in the experimental studies, where dietary dosage was 5 ppm. One specimen contained 7 ppm. Quantities of dieldrin were 1 ppm or lower in 12 specimens, 2 ppm in 1 specimen, and 8 ppm in 1 specimen.

The eagle with 7 ppm DDT + DDD in the brain also contained 8 ppm dieldrin. Quantities of DDT + DDD above 30 ppm in the brain can be considered in a zone of serious concern, as discussed above, but this amount is considerably above 7 ppm. However, 8 ppm of *dieldrin probably is more critical*, as indicated by residues in animals found dead in the field and in those killed in laboratory studies. Brain residues in 23 animals found dead after field applications of dieldrin ranged from 2 to 20 ppm, with only one below 6 ppm. These animals included meadowlarks, cottontail rabbits, and cotton rats (Patuxent Center, unpublished data); green-winged teal, redhead duck, lesser scaup, and shoveller (Sheldon *et al.*, 1963) with no evidence to suggest species differences. Six domestic animals (cattle, sheep, and dogs) dosed experimentally contained 10-30 ppm of dieldrin in their brains (Kitselman *et al.*, 1950). Two experimentally dosed pheasants died containing 10 and 18 ppm of dieldrin in their brains (McEwen *et al.*, 1963) and six dogs that died or were killed *in extremis* contained 2 to 9 ppm (average, 6 ppm) (Harrison *et al.*, 1963). Thus, 8 ppm must be considered in the zone of hazard.

This particular eagle was an immature female, found dead at the base of a known roosting tree near Vernon, Vermont, on May 1, 1964. Autopsy by L. N. Locke showed the bird to be very thin, but without diagnostic lesions.

Earlier analyses of field eagles made at our laboratory by colorimetric methods of Schechter *et al.* (1945) and read at wavelength 540 showed residues of DDE overshadowing whatever DDT + DDD may have been present. These DDE residues were between 0 and 8 ppm in the brains of 22 eagles, about 17 ppm in 1, and between 31 and 35 ppm in 5. With DDT and DDD present in smaller, although unknown, amounts, it is apparent that in this group also the amounts were below the lethally critical zone in most. The significance of the five readings above 30 ppm cannot be properly evaluated.

DDE residues in brains of the eagles read for the spectrum of pesticides were less than 2 ppm in 10 specimens, 6 to 33 ppm in 4 specimens. The 33 ppm occurred in the same eagle that had high readings of DDT + DDD and dieldrin. Quantities of DDE in the brain ordinarily were higher than those of DDT + DDD but occasionally approached equality.

Liver residues also will be given here for the record. In the

broad spectrum analyses, the livers of 16 birds contained traces to 42 ppm of DDE with a median value of 2 ppm. Twelve contained less than 10 ppm. DDT + DDD measured from traces to 8 ppm, with a median of 1 ppm. Dieldrin measured 1 or <1 ppm in 14 specimens, 2 ppm in 1 specimen, and 5 ppm in one specimen. The DDE residues determined earlier by colorimetric methods in 61 specimens ranged from none detected to 305 ppm, with a median value of 6 ppm. The higher median value in the colorimetric series may have been primarily the result of methodological differences, for comparisons have shown that colorimetric readings made at the DDE wavelength generally are somewhat higher than DDE readings by gas and thin layer chromatography.

Measurement of the *quantity* of pesticides stored in the body is useful as an indicator of environmental contamination, and of the reserve supply that may become critical when food supply is reduced or weight is lost for other reasons. For this purpose, we have analyzed the carcass remainders, which include the entire body after the gastrointestinal tract, brain, and liver have been removed. The quantities of DDE in 16 specimens ranged from traces to more than 50 ppm with a median of 9 ppm. Trace quantities of heptachlor epoxide were present in about half the specimens. The most nearly comparable readings from the earlier colorimetric analyses are those for muscle. In these, DDE ranged from 0 to 118 ppm in 61 specimens with a median of 5 ppm.

Nine bald eagle eggs have been analyzed for pesticide residues (Table 3). Residue readings were adjusted to permit comparisons, for different amounts of drying in over-age eggs are a source of great

TABLE 3. PESTICIDES IN EGGS OF BALD EAGLES

Date	Location	Parts Per Million (as weighed at analysis)				Parts Per Million (adjusted)[1]			
		DDE	DDD	DDT	Dieldrin	DDE	DDD	DDT	Dieldrin
1962	New Jersey[2]	30.0	—	—	—	23-32	—	—	—
1962	New Jersey[2]	11.1	—	—	—	4-6	—	—	—
1962	New Jersey[2]	21.3	—	—	—	11-14	—	—	—
1963	Missouri[2]	5.6	—	—	—	4-6	—	—	—
1963	Missouri[2]	1.1	—	—	—	1	—	—	—
1964	Maine	11.9	2.5	0.2	2.5	4.6	0.8	0.1	0.8
1964	New Jersey	8.6	8.6	0.3	1.7	4.6	4.6	0.2	0.9
1964	New Jersey	7.6	0.1	0.3	1.5	5.0	4.0	0.2	1.0
1965	Florida[3]	21.5	4.3	0.0	0.0	13.0	2.6	0.5	0.5

[1] Adjustment procedure is described in the text.
[2] *Colorimetric readings, primarily* DDE.
[3] Also a trace of heptachlor epoxide.

distortion if weights are taken at face value. The adjustments were made on the basis of egg volume as described by Stickel *et al.* (1965). The specific gravity was taken as 1 mg per ml, since the actual specific gravity of fresh eagle eggs is not accurately known. Adjusted residue readings (primarily DDE) in five eggs analyzed colorimetrically ranged from 1 to about 30 ppm. Four read for a broader spectrum of compounds contained 5 to 13 ppm of DDE, 1 to 5 ppm of DDT + DDE and 0.5 to 1 ppm of dieldrin.

Transmission of pesticide residues from parent to offspring via the eggs of birds is well known. The quantity of these residues that may indicate an adverse effect on hatching and survival is far from clear, however. Genelly and Rudd (1956) have reported that pheasant eggs containing 162 and 349 parts per million hatched as well as the controls, but that early survival was reduced. Keith (1965) reported 202 ppm DDE, 19 ppm DDT, and 6 ppm DDD in live gull eggs. Quantities in eagle eggs so far reported are so much lower than these that they provide little basis for surmising that DDT in the eggs prevented hatching. Interpretation of the effects of dieldrin or of dieldrin plus the DDT-related compounds cannot presently be made.

The conclusions to be drawn from the results of field analyses are, first, that exposure of eagles to DDT and dieldrin is nationwide, as it is for most other animals, including people; second, that at least an occasional eagle obtains enough dieldrin and possibly enough DDT to place him in hazard; and third, that most eagles that die in the United States today die of causes other than pesticide poisoning. The important question of sublethal effects on behavior, particularly parental behavior, cannot yet be answered.

RESEARCH PLANS

Our plans for further work with eagles include a continuous monitoring of eggs and adults for pesticide residues. We hope to extend these analyses to include some of the more important heavy metals that are present as environmental pollutants, for we view the eagle problem as a part of the larger problem of environmental pollution that affects many species and many environments. Food chain investigations specific to eagles will be undertaken jointly with the National Audubon Society, as part of our research in ecological systems.

We believe the need for better understanding of the meaning of residues in eggs and tissues remains critical, and will require additional experimentation with various species. It has seemed important to make tests with a predatory species, even if it could not be the

eagle. Hence we have established a colony of sparrow hawks to test reproductive effects. Thirty-six pairs are now on experiment with dosages of DDT and dieldrin.

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